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Fatal body positioning during epidural anesthesia in a ewe

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Epidural anesthesia is useful for intra-abdominal, pelvic or hindlimb surgery in sheep because it is inexpensive, easy to perform, and provides good analgesia (1,2,3). However, transient convulsions (4,5), persistent bilateral hindlimb paralysis (6), and unexplained deaths (7) have been reported following use of the technique. We describe herein the death of a ewe during epidural anesthesia; the death was probably caused by unsuitable body position.

A four-year-old pregnant cross-bred ewe, which had been recently purchased by the college for use in research, was referred to the teaching hospital because of dystocia. The animal, which weighed 95 kg, was weak in the hindlimbs and had difficulty standing without assistance. Clinical examination did not reveal the cause for this. No other abnormalities were found and because fetal oversize was diagnosed, cesarean section was performed.

Premedication was xylazine (Rompun, Haver, Shawnee, Kansas) given intra-muscularly at a dose of 0.05 mg/kg. When sedation was apparent ten minutes later, a catheter was placed in the cephalic vein and lactated Ringer's solution was infused at 10 mL/kg/h. The skin over the lumbosacral area was clipped and surgically prepared. With the patient in lateral recumbency, the spine was flexed and the dorsal process of the last lumbar vertebra was identified lying beneath an imaginary line joining the iliac crests. A 20-gauge 8 cm spinal needle with stylet was introduced in the midline immediately caudal to this process and was slowly advanced until the ligamentum flavum had been penetrated. The accurate position of the needle tip in the epidural space was indicated by the failure to aspirate cerebrospinal fluid and the lack of resistance offered to an injection of sterile saline. Eight mL of 2% lidocaine solution without epinephrine

(Lid-o-cain, Butler, Columbus, Ohio), which had been warmed to body temperature, was then injected over 30 seconds. The ewe was then held on her back for five minutes while the left sublumbar fossa was prepared for surgery. The pulse rate, rhythm and quality were monitored throughout anesthesia by palpation of the median artery, while respiratory rate and depth were assessed visually. Throughout surgery (during which live twin-lambs were delivered) the patient was positioned in right lateral recumbency and the heart and respiratory rates were stable. During closure of the abdominal wall, however, the ewe struggled and the pulse rate increased from 85 to 125 beats per minute, becoming weaker. This suggested that analgesia was waning, so 4 mL 2% lidocaine solution was given using the technique described previously. This time, however, the ewe was kept in right lateral recumbency. The animal became calm, the heart rate fell to previous levels, and surgery was completed without further problems.

This pooling of blood probably resulted from the hypotensive effects of the epidural anesthetic being enhanced by the peculiar body position after milking

Ten minutes after the second injection, the ewe was rolled into sternal recumbency and the hindlimbs and udder were slid off the operating table in order to collect colostrum. The ewe was alert and responded to the lambs. Throughout milking, the body was supported horizontally, but when milking was completed five minutes later, the hindquarters were allowed to fall below the level of the table. In two minutes the sheep was unconscious; her eyes were open, but palpebral and corneal reflexes were absent. Breathing was not obvious and the pulse was weak and fast. The animal was immediately repositioned on the table and fluids were infused at the maximum possible rate. Despite this action, the heart-sounds grew inaudible, the pulse quality deteriorated and the ewe was pronounced dead.

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Gross postmortem examination performed 12 hours later revealed that the splanchnic and pelvic viscera and the muscles of the hindlimbs were congested. This congestion was not present in the thoracic viscera or in the muscles of the forelimbs and neck. Gross examination of the spinal cord did not reveal the cause of hindlimb weakness observed at presentation. The course of events and pathological findings suggest that death was related to blood pooling in the hindquarters and pelvic viscera. This pooling of blood probably resulted from the hypotensive effects of the epidural anesthetic being enhanced by the peculiar body position after milking.

Following epidural injection, local anesthetics can cause hypotension in several ways (8), but primarily through inhibition of preganglionic sympathetic B fibers which mediate vasoconstriction in vascular smooth muscle. This results in vasodilation, increased blood vessel capacitance and reduced vascular resistance in tissues innervated by affected nerves. In man, arterial hypotension results from decreased cardiac output secondary to blood pooling in dilated veins, while arteriolar dilation with decreased systemic vascular resistance contributes minimally (9). The extent of spread of local anesthetics in the epidural space is the principal factor in the development of systemic hypotension (10), and this is related to the volume of drug injected and the dimensions of the epidural space. Because sympathetic B fibers are more sensitive to local anesthetics than the more heavily myelinated and larger A α (motor), A β (touch, pressure), and A δ (pain, temperature) fibers (11), autonomic blockade may exist in spinal segments in which motor and sensory function is unaffected. In other words, after epidural lidocaine injection, sympathetic paralysis extends more cranially than is indicated by analgesia or muscle relaxation.

Despite the adverse consequences of excessive sympathetic blockade, the doses of lidocaine recommended for epidural anesthesia in the sheep vary considerably. Hall and Clark (1) suggest that 8 to 15 mL of 1.5% solution be used, depending on the size of the sheep. Others recommend substantially greater doses of 1 mL of 2% solution per 4.5 kg (3) or per 5.0 kg (2). The initial dose used in the case described (1 mL 2% solution per 12 kg) falls between these recommendations. When the second injection (1 mL 2% solution per 24 kg) is added the total dose still remains less than some recommendations (2,3). In pregnant women, there is an exaggerated response to epidural anesthesia because the longitudinal vertebral venous sinuses become engorged with blood and reduce the volume of the epidural space (12). Under these conditions, a given injection volume extends more cranially and increases the likelihood of sympathetic paralysis (8). Total spinal block, including sympathetic paralysis also occurs when normal doses of anesthetic are injected into the subarachnoid space (13). This occurs because the drug diffuses more rapidly and cranially through cerebrospinal fluid than epidural fat. Inadvertent subarachnoid injection is possible at the lumbosacral junction in sheep because the spinal cord and meninges extend into the midsacral (S2-S3) region (14). However, this potential complication is easily recognized and there-

fore avoided because arachnoid puncture is indicated by the free flow of cerebrospinal fluid from the needle hub (15). In the case described here, it seems unlikely that the combined doses used were excessive or that inadvertent subarachnoid injection had occurred because cardiovascular changes were not seen within fifteen minutes of the second injection. It is probable that when the hindquarters were below table level, blood rapidly pooled in the dilated vessels of the pelvic viscera and hindlimb musculature. Hypotension then occurred because venous return (and therefore cardiac output) was reduced, while systemic vascular resistance was lowered. Arterial pressure probably fell below the range compatible with adequate myocardial perfusion, in which case death resulted from myocardial ischemia.

Although epidural anesthesia is a useful technique and is easily performed in sheep, its use does not obviate the need for careful patient management

Under normal circumstances, blood pressure would have been maintained by compensatory increases in systemic vascular resistance and cardiac output. While reflex tachycardia was observed in this patient, full compensation was unlikely, being impaired in at least three ways. Principally, vasoconstrictor fibers to caudal segments were blocked by the epidural anesthetic. Second, myocardial and peripheral vascular responsiveness to endogenous catecholamines was probably altered by the xylazine premedication. While the dose used in this patient (0.05 mg/kg intramuscularly) is recommended in sheep (16) the drug causes bradycardia and arterial hypotension in other species (17,18). A third possible factor limiting the animal's ability to compensate for hypotension is linked to the cause of the preoperative hindlimb weakness. A space-occupying lesion in the spinal cord, for example, may have reduced the volume of the epidural space causing a given volume of injectate to extend more cranially, increasing the extent of sympathetic paralysis. Alternatively, spinal cord pathology causing lower motor neuron disease and muscle weakness may have affected sympathetic vasoconstrictor fibers. Unfortunately, gross spinal cord pathology was not found on postmortem examination. It is possible that a metabolic disease such as hypocalcemia was obtunding reflex cardiovascular responses to hypotension, but this is also unlikely; the preoperative clinical examination did not reveal abnormalities other than hindlimb weakness.

The sheep was not dehydrated before surgery, but fluids were given in anticipation of hemorrhage and anesthesia-related hypotension. The infusion rate used (10 mL/kg/h) was insufficient, however. In pregnant women, 500 to 2000 mL of dextrose-free crystalloid is infused prophylactically before epidural anesthesia; the absolute volume depending on the intended level of blockade (19). This strategy has not been recommended in the veterinary literature which is unfortunate; adequate volume preloading would probably

have postponed the onset of fatal hypotension. Because sheep approximate human size, similar volumes of intravenous fluid are probably required.

After the ewe collapsed, three responses other than rapid fluid infusion may have changed the outcome of this case. First, raising the animal's hindquarters above the level of the heart would have prevented blood pooling in this area and improved venous return. Second, the trachea should have been intubated and respiration supported with positive pressure ventilation and oxygen. Third, α_1 adrenergic agonist drugs such as ephedrine, phenylephrine, methoxamine or metaraminol may have been given in order to constrict the dilated vascular beds. Ephedrine is particularly useful in this situation because, compared with other α_1 agonists, it preserves uterine blood flow in the pregnant ewe (20). The routine use of metaraminol (5 mg intramuscularly) during epidural anesthesia in sheep has been recommended and if hypotension is severe, it is advised that 5–10 mg methoxamine be given intravenously (1).

Although epidural anesthesia is a useful technique and is easily performed in sheep, its use does not obviate the need for careful patient management. Patient positioning, for example, must be considered in order to avoid severe and occasionally fatal hypotension.

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